

IMAGING

CLINICAL CASE SERIES

Tuberculous Constrictive Pericarditis in Immunocompetent Migrants From High-Incidence Countries



A Conservative Approach in a Low-Incidence Setting

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ABSTRACT

BACKGROUND Although Italy is a low-incidence country, global migration patterns necessitate consideration of tuberculosis (TB), particularly extrapulmonary forms, in the differential diagnosis.

CASE PRESENTATIONS We report 2 cases of tuberculous pericarditis in young, immunocompetent, HIV-negative men from high TB-incidence regions who were hospitalized at the Infectious Diseases Unit of Policlinico Umberto I, Rome. In both patients, the diagnosis of TB was made presumptively based on clinical presentation, positive immunological markers, and characteristic findings on multimodal imaging. Both patients had clinical and imaging evidence of constrictive pericarditis. A conservative medical approach was initiated, combining standard anti-TB therapy with prednisone 60 mg daily. This regimen led to significant clinical and hemodynamic improvement. One patient's course was complicated by a rare intracardiac tuberculoma, which also regressed with therapy.

CONCLUSIONS Our cases demonstrate that a prompt, conservative approach to tuberculous pericarditis, confirmed by functional recovery and imaging findings, can lead to marked clinical and hemodynamic improvement, potentially obviating the need for invasive procedures or surgical intervention in selected patients. (JACC Case Rep. 2026;31:106413) © 2026 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

BACKGROUND

Tuberculosis (TB) remains a major global health threat. According to the World Health Organization, the estimated global burden of TB is steadily increasing, with most cases reported in the Southeast Asia and African regions.¹ Although Italy remains a

low-incidence country, global migration patterns necessitate that TB, particularly extrapulmonary forms, be considered in the differential diagnosis.

Although pulmonary involvement is the most common presentation of TB, dissemination can affect any organ system.² Cardiovascular involvement, while uncommon, accounts for up to 5% of cases in

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**ABBREVIATIONS
AND ACRONYMS**

CT = computed tomography
MRI = magnetic resonance
imaging
RIPE = rifampicin, isoniazid,
pyrazinamide, ethambutol
TB = tuberculosis

immunocompetent individuals and is a leading cause of constrictive pericarditis in endemic regions.³ The clinical presentation of tuberculous pericarditis can range from acute pericarditis to advanced heart failure.⁴

In this case series, we describe 2 immunocompetent patients with tuberculous pericarditis who were recently managed at the Infectious Diseases Unit of Policlinico Umberto I in Rome. These cases highlight the heterogeneity of the disease's presentation, the challenges of presumptive diagnosis, and the potential efficacy of a conservative therapeutic approach, with particular emphasis on the role of corticosteroid therapy.

CASE 1

HISTORY OF PRESENTATION. A 28-year-old man from India, living in Italy since 2022, presented to the emergency department of Policlinico Umberto I with a 1-month history of fever, fatigue, and unintentional weight loss. He had a prior hospitalization 3 months earlier for acute pericarditis, which was managed conservatively with nonsteroidal anti-inflammatory drugs and colchicine. On admission, laboratory tests revealed elevated C-reactive protein. Transthoracic echocardiography showed a mild circumferential pericardial effusion with preserved left ventricular function. Given the persistent febrile illness of unknown origin, the patient was admitted to the infectious disease unit for further investigation.

INVESTIGATION. During the early days of hospitalization, he developed new-onset atrial fibrillation with rapid ventricular response, which

TAKE-HOME MESSAGES

- A high index of suspicion for tuberculous pericarditis is essential, especially in low-incidence countries with a high migrant population.
- A prompt, conservative approach combining standard antituberculosis therapy with high-dose corticosteroids can lead to marked clinical and hemodynamic improvement in carefully selected patients.

spontaneously reverted to sinus rhythm. Serial electrocardiograms and echocardiograms confirmed the persistence of a nontamponading pericardial effusion, with preserved biventricular systolic function (left ventricular ejection fraction: 60%) and a mild septal bounce. Contrast-enhanced chest computed tomography (CT) revealed marked concentric pericardial thickening (up to 23 mm) and mediastinal lymphadenopathy. Although sputum tests for acid-fast bacilli and *Mycobacterium tuberculosis* polymerase chain reaction were negative, both the Mantoux test and interferon gamma release assay yielded strongly positive results. HIV testing was negative. Empirical anti-TB therapy with a rifampicin, isoniazid, pyrazinamide, and ethambutol (RIPE) regimen was initiated. Positron emission tomography (PET)-CT confirmed active inflammation with intense FDG (¹⁸F-fluorodeoxyglucose) uptake in the thickened pericardial layers (maximum standardized uptake value: 22.5) and mediastinal lymph nodes, highly suggestive of tuberculous pericarditis. Cardiac magnetic resonance imaging (MRI) (Figure 1A) confirmed severe pericardial thickening (up to 5 mm),

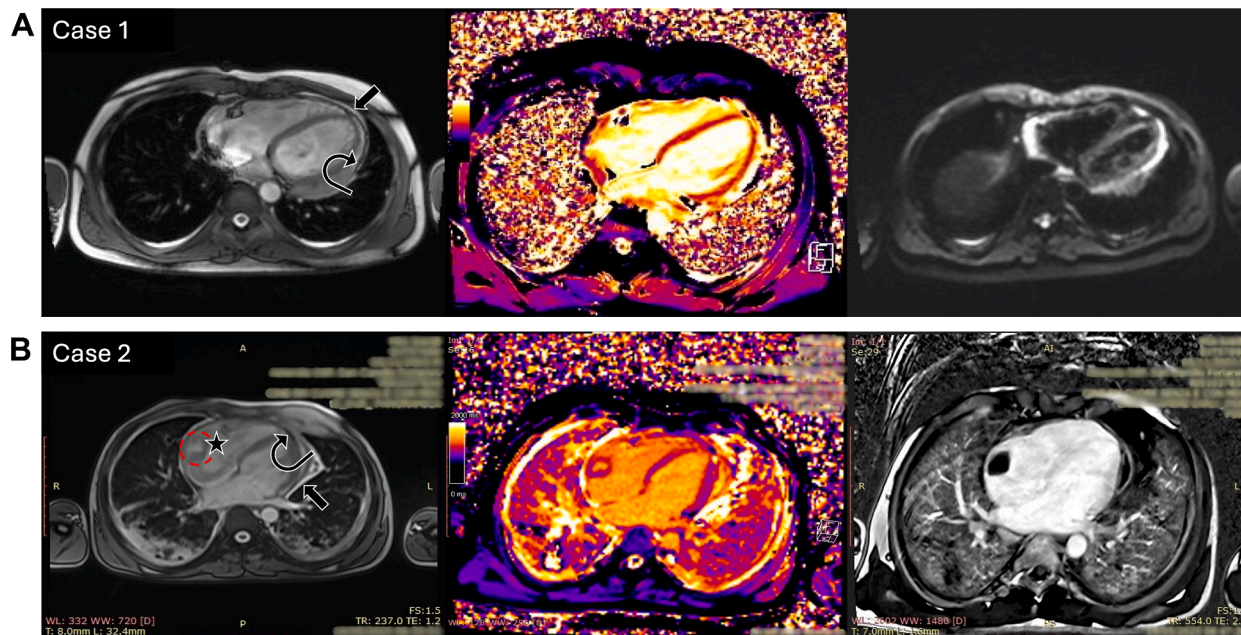
VISUAL SUMMARY Timeline of Clinical Management Comparing Cases 1 and 2

Case 1		Case 2	
Day 0	Empirical antituberculosis therapy (RIPE regimen) initiated after both Mantoux test and IGRA yielded strongly positive results.	Day 0	Antituberculosis therapy (RIPE regimen) initiated after diagnosis of disseminated tuberculosis with involvement of the lungs, pleura, abdominal lymph nodes, and kidneys.
Day 21	High-dose prednisone therapy (60 mg daily) started after PET-CT and cardiac MRI findings consistent with probable tuberculous pericarditis.	Day 42	High-dose prednisone therapy (60 mg daily) started after cardiac MRI findings consistent with probable tuberculous pericarditis.
Day 41	Discharged from hospital with outpatient follow-up scheduled.	Day 56	Transitioned to maintenance phase of antituberculosis therapy with isoniazid and ethambutol.
Day 49	Prednisone tapered to 30 mg daily after echocardiographic confirmation of preserved global cardiac function.	Day 64	Discharged from hospital with outpatient follow-up scheduled.
Day 56	Transitioned to maintenance phase of antituberculosis therapy with isoniazid and ethambutol.	Day 84	Missed scheduled outpatient visit for re-evaluation and corticosteroid tapering.
Day 77	Prednisone further tapered to 15 mg daily after repeat cardiac MRI and cardiothoracic evaluation.		
Day 91	Next outpatient follow-up scheduled.		

Chronological summary of diagnostic and therapeutic milestones in the 2 cases.

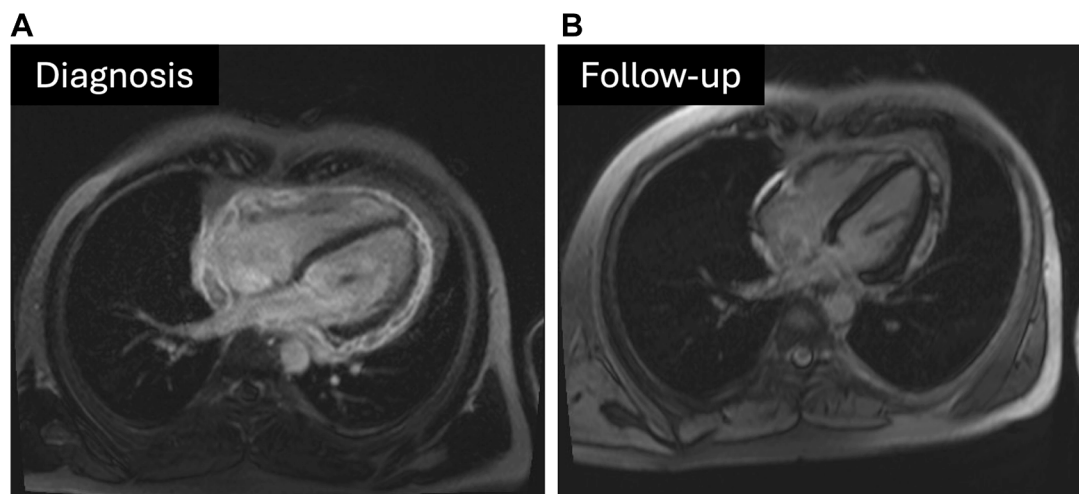
IGRA = interferon gamma release assay; MRI = magnetic resonance imaging; PET-CT = positron emission tomography-computed tomography; RIPE = rifampicin, isoniazid, pyrazinamide, ethambutol.

FIGURE 1 Comparison of Cardiac MRI Between Cases 1 and 2



(A) In case 1, cardiac MRI showed severe pericardial thickening (straight arrows) with associated edema (curved arrows) and adhesions. (B) In case 2, cardiac MRI revealed massive concentric pericardial thickening (straight arrows) with associated edema (curved arrows), biatrial dilatation, and a sessile right atrial mass (star), consistent with a probable tuberculoma. Overall, the findings supported the diagnosis of constrictive pericarditis of probable tuberculous etiology. MRI = magnetic resonance imaging.

FIGURE 2 Comparison of Cardiac MRI Between Diagnosis and Follow-Up for Case 1



Comparison between cardiac MRI (A) at diagnosis and (B) after 4 weeks of treatment shows persistent pericardial thickening and edema, fibrous adhesions, and a mildly reduced, fluid-corpused pericardial effusion. MRI = magnetic resonance imaging.

associated edema, and adhesions, with mildly reduced biventricular systolic function (left ventricular ejection fraction: ~50%). These findings were consistent with constrictive pericarditis of probable tuberculous etiology.

MANAGEMENT AND OUTCOME. Given the absence of hemodynamic compromise, intracardiac catheterization and pericardiocentesis was deferred. After a cardiology consultation, a conservative management plan was adopted, and high-dose prednisone (60 mg daily) was initiated. The patient remained clinically stable throughout hospitalization, with no signs of tamponade, peripheral edema, or drug-related adverse effects. He was discharged in good condition, with scheduled outpatient follow-up.

FOLLOW-UP. After 4 weeks of corticosteroid therapy, the dosage was tapered to prednisone 30 mg daily after echocardiographic confirmation of preserved global function. A follow-up cardiac MRI after 8 weeks of conservative therapy (Figure 2) revealed persistent pericardial thickening and edema with fibrous adhesions, a mildly reduced fluid-corporuscled pericardial effusion, and preserved biventricular systolic function. After cardiothoracic evaluation, the prednisone dose was further tapered to 15 mg daily. The patient remained completely asymptomatic (NYHA functional class I) while continuing corticosteroids and anti-TB therapy, with ongoing clinical monitoring and a planned reassessment for additional tapering.

CASE 2

HISTORY OF PRESENTATION. A 21-year-old man from Bangladesh, living in Italy since 2023, was diagnosed with disseminated TB based on positive acid-fast bacilli in sputum and urine. His disease involved lungs, pleura, abdominal lymph nodes, and kidneys. He was started on a standard RIPE regimen. HIV testing was negative. The clinical course was complicated by persistent tachycardia and conduction abnormalities (P pulmonale, T-wave inversion), which prompted further cardiologic evaluation. Transthoracic echocardiography showed preserved biventricular function (left ventricular ejection fraction: ~60%).

INVESTIGATION. Although his initial echocardiography showed preserved function, a follow-up study after 3 weeks revealed a marked reduction in biventricular systolic function (left ventricular ejection fraction: 35%/40%) owing to global hypokinesia, with evidence of septal bounce but without evidence of pericardial effusion, confirmed by transesophageal

echocardiography, which also detected a solid mass within the right atrium. Cardiac MRI (Figure 1B) identified massive concentric pericardial thickening (up to 16 mm), biatrial dilatation, and a sessile right atrial mass (50 × 24 mm), consistent with a probable tuberculoma. Coronary angiography excluded significant stenoses but demonstrated slow contrast runoff in the left coronary artery, supporting the presence of hemodynamic compromise. The patient was admitted to the intensive cardiac care unit, where cardiothoracic surgery was consulted; however, urgent pericardiectomy was deferred in favor of conservative management given the complete absence of symptoms and the recent onset of the condition. Invasive hemodynamic assessment was not performed, as echocardiography and MRI provided clear noninvasive evidence of constrictive physiology. These findings were consistent with pericardial involvement of probable tuberculous etiology, and the patient was subsequently transferred to the infectious disease unit.

MANAGEMENT AND OUTCOME. After the MRI, high-dose prednisone (60 mg daily) and anticoagulation were initiated. With intensified medical therapy, the patient's condition improved significantly. He was discharged in stable condition, with improved left ventricular ejection fraction (up to 55%), stable pericardial effusion, and reduction in the size of the right atrial tuberculoma (now 36 × 21 mm). At discharge, he continued anti-TB therapy, high-dose corticosteroids, and anticoagulation.

FOLLOW-UP. A repeat cardiac MRI was scheduled after discharge, and corticosteroids were to be tapered to 30 mg daily after 4 weeks of high-dose therapy. Despite multiple attempts to contact him, the patient failed to attend scheduled outpatient follow-up visits, underscoring the challenges of ensuring long-term adherence and monitoring in migrant populations affected by TB. Two months after discharge, colleagues from a hospital in another Italian region contacted us seeking clinical information about the patient. They confirmed that he was still taking his prescribed medications as indicated at discharge and that his left ventricular ejection fraction remained stable. We subsequently spoke directly with the patient and attempted to schedule a new follow-up visit.

DISCUSSION

These cases highlight the diagnostic and therapeutic challenges of tuberculous pericarditis, especially in low-incidence settings. In both patients, the

TABLE 1 Diagnostic Criteria for Tuberculous Pericarditis	
Definite (or proven) TBP	- Isolation of <i>Mycobacterium tuberculosis</i> bacilli from pericardial fluid or tissue samples; and/or - Positive PCR for <i>M tuberculosis</i> in pericardial fluid or tissue; and/or - Histological evidence of caseating granulomas in pericardial tissue.
Probable (or presumed) TBP	- Suggestive pericardial fluid with lymphocytic, exudative characteristics and elevated adenosine deaminase; and/or - Evidence of tuberculosis at another extrapulmonary site; and/or - Positive immunologic test for tuberculosis; and/or - Good clinical and/or radiologic response to antituberculosis therapy.
Possible (or plausible) TBP	Pericardial disease of uncertain etiology, without microbiological or histological confirmation, in which tuberculosis remains a plausible but unconfirmed cause based on clinical, epidemiological, and imaging findings.

The diagnosis of tuberculous pericarditis is considered definite (or proven) when *M. tuberculosis* is demonstrated in pericardial fluid or tissue; probable (or presumed) when there is no direct microbiological confirmation but strong clinical and/or imaging evidence supported by immunologic or systemic findings; and possible (or plausible) when pericardial effusion or constrictive pericarditis occurs without direct microbiological or histological evidence, yet tuberculosis remains a plausible but unconfirmed cause based on clinical, epidemiological, and imaging data. Both cases in this series met the criteria for probable (or presumed) tuberculous pericarditis.

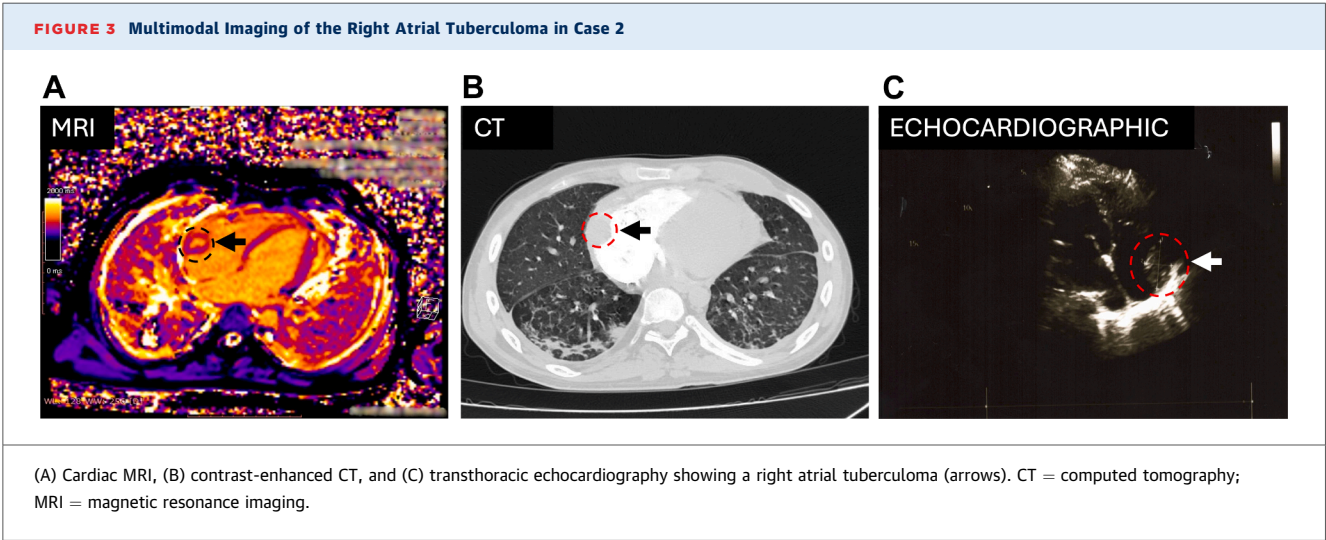
PCR = polymerase chain reaction; TBP = tuberculous pericarditis.

diagnosis was presumptive, lacking microbiological confirmation from pericardial fluid or tissue, a common limitation in this paucibacillary condition.⁵ Indeed, even in endemic regions, the sensitivity of pericardiocentesis or biopsy rarely exceeds 70%, and culture results can take weeks, making clinical decision-making difficult. For this reason, multimodal imaging has emerged as an essential tool. The diagnosis was established through a combination of clinical suspicion, strong immunological responses, and multimodal imaging leading to the classification of both cases as probable (or presumed) tuberculous pericarditis. The diagnostic criteria applied are summarized in Table 1. Cardiac MRI was instrumental in confirming the presence of severe pericardial thickening and constrictive physiology,⁶ while PET-CT provided evidence of active inflammation, supporting the diagnosis of an active tuberculous process.⁷ Of note, the intracardiac tuberculoma in case 2 is a

rare finding that responded favorably to medical therapy, further supporting the presumptive diagnosis (Figure 3).

Both patients were treated with a standard RIPE regimen for the initial 2 months, after which continuation with isoniazid and ethambutol for an additional 7 months was scheduled in consideration of the pericardial involvement, for a total planned treatment duration of 9 months.

Our experience supports the use of corticosteroids as an adjunctive treatment for tuberculous pericarditis. Despite mixed evidence regarding mortality, studies suggest that corticosteroids can reduce the incidence of constrictive pericarditis and hasten symptom resolution, particularly in HIV-negative patients.^{8,9} In both of our immunocompetent patients, the prompt initiation of high-dose corticosteroids in combination with anti-TB therapy resulted in a marked reversal of cardiac dysfunction, potentially



obviating the need for invasive procedures. Nevertheless, corticosteroid use requires careful balance: although they may reduce inflammation and improve hemodynamics, they also carry risks of immunosuppression, metabolic complications, and masking of disease progression. In line with previous reports, both patients were managed with the following corticosteroid tapering regimen: prednisone 60 mg daily for the first 4 weeks, 30 mg daily for weeks 5 to 8, 15 mg daily for weeks 9 to 10, and 5 mg daily for week 11. To ensure safe tapering and treatment monitoring, laboratory tests (complete blood count, C-reactive protein, creatinine, and liver enzymes) and infectious disease evaluations were scheduled at weeks 4, 8, and 10. In addition, follow-up transthoracic echocardiography with cardiologic assessment was planned at week 4, and a repeat cardiac MRI with cardiothoracic surgical consultation at week 8. Upon completion of corticosteroid tapering, a bimonthly clinical and laboratory follow-up was planned to monitor for late complications and ensure adherence to anti-TB therapy. In case 1, the reduction in inflammatory markers, stable hemodynamic compensation on echocardiography, and partial resorption of the fluid-corpusculated pericardial effusion observed on follow-up MRI were considered reliable indicators to proceed with corticosteroid tapering. In case 2 however, loss to follow-up prevented further data collection and evaluation of treatment response.

The successful nonsurgical management of constrictive pericarditis in these patients aligns with emerging evidence that the condition may be reversible with medical therapy, especially when initiated during the subacute inflammatory phase. Case 1 demonstrated the utility of a strictly conservative approach with close clinical and imaging follow-up, which allowed stabilization without surgical intervention. Conversely, case 2 underlined how rapid clinical deterioration and the presence of a rare complication such as an intracardiac tuberculoma can still be managed effectively with tailored medical therapy. In this context, imaging findings and clinical stability played a pivotal role in distinguishing the inflammatory from the fibrotic phase of constriction. The absence of extensive calcification or fibrotic scarring on cardiac MRI, together with the preserved hemodynamic stability of both patients, supported the decision to defer surgery in favor of a conservative approach. In case 1, MRI revealed circumferential pericardial thickening with clear evidence of pericardial edema and enhancement, and absence of late gadolinium enhancement. In case 2,

MRI demonstrated concentric pericardial thickening with limited calcifications, and areas of nonischemic late gadolinium enhancement. Importantly, there was no evidence of dense adhesions or extensive fibrosis, and both ventricles retained the potential for functional recovery. Altogether, these features indicated a subacute inflammatory phase of constrictive pericarditis, explaining the favorable response to medical therapy and supporting the rationale for a conservative approach.

Another key aspect emerging from our report is the importance of continuity of care. The partial loss to follow-up of case 2 illustrates how migrant populations are at higher risk of treatment interruption owing to socioeconomic vulnerability, language barriers, and limited health system integration. The effectiveness of even the best treatment plan can be compromised by a patient's loss to follow-up, a critical issue that requires structured discharge planning and culturally sensitive community outreach. Addressing these challenges through multidisciplinary coordination, community-based support, and tailored follow-up programs is crucial to ensure long-term treatment success and to reduce the risk of relapse or progression.¹⁰

Overall, our findings demonstrate that in selected patients, tuberculous constrictive pericarditis can be managed conservatively, provided there is early recognition, access to multimodal imaging, and structured follow-up. This approach may help avoid unnecessary pericardiectomy in the acute phase, reserving surgery for cases with persistent constriction despite optimized medical therapy.

CONCLUSIONS

Early recognition and prompt management of tuberculous pericarditis are critical for patient outcomes. TB should be considered in the differential diagnosis of pericardial disease in patients from high-prevalence settings, even in low-incidence countries. Advanced imaging modalities can facilitate timely diagnosis. Our cases provide additional evidence that supports a conservative approach combining standard anti-TB therapy with high-dose corticosteroids. This strategy can achieve significant clinical and hemodynamic improvement and may represent a reasonable alternative to pericardiectomy in carefully selected patients with evidence of a reversible, inflammatory phase of constrictive pericarditis, who can be closely monitored with reliable follow-up.

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